cancer. In this series it was a more constant finding in gastric extracts than were absent free hydrochloric acid, the presence of lactic acid, and the glycyltryptophan test. It was rather more constant than tests for occult blood and the demonstration of gastric motor inefficiency. It was not so consistent in its manifestation as the demonstration of organisms of the Boas-Oppler group or the increase in the formol index.

2. In extragastric malignancy, gastric syphilis, and nephritis the Wolff-Junghans' test is inconstant.
3. In the differentiation between malignant and non-malignant achylias the Wolff-Junghans' test, when interpreted in connection with other clinical and laboratory data, is of considerable value. Positive reactions are rarely obtained in the achylias of primary anemia, simple achylia gastrica, and simple achlorhydrias when such are unassociated with gastric motor inefficiency.
4. Simple gastric and duodenal ulcers, especially when accompanied by pyloric stenosis or gastric atony, may give confusing responses to the Wolff-Junghans' test.
5. The presence of blood in gastric extracts may be a factor in the production of certain atypical positive tests.

JAUNDICE IN TUBERCULOSIS.

By John M. Cruice, A.B., M.D.

PHYSICIAN TO THE HENRY PHIPPS INSTITUTE OF THE UNIVERSITY OF PENNSYLVANIA, AND TO THE OUT-PATIENT DEPARTMENT OF ST. AGNES' HOSPITAL; INSTRUCTOR IN MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, AND MEMBER OF THE JOHN MORGAN SOCIETY.

(From the Henry Phipps Institute of the Univerity of Pennsylvania.)

Jaundice is caused by the presence of bile pigment in the blood. It is now acknowledged that the passage of the bile pigment is by way of the lymphatics into the thoracic duct and thus into the general circulation, and is always caused by some obstruction, whether extrahepatic or intrahepatic, to the normal flow of the bile. Rolleston divides jaundice into: "(1) Extrahepatic or 'obstructive' where there is a gross obstruction, usually involving the larger extrahepatic bile ducts, to the flow of bile along the bile ducts. (2) Toxemic, intrahepatic, or hemohepatogenous, where there is obstruction in the small intrahepatic bile ducts. The obstruction is due to cholangitis or inflammation of the minute ducts, depending upon the irritating effect of poisons derived from the blood circulating through the liver." In tuberculosis we find jaundice caused by both extrahepatic and intrahepatic obstruction to the flow of bile.

The extrahepatic cases have been caused by tuberculous glands in the portal fissure pressing upon the bile ducts. This is a rare condition, from the fact that the glands in the portal fissure receive the efferent lymphatic vessels conveying the lymph away from the liver and not those conveying lymph from the intestines and peritoneum. These glands become infected through the liver, which has become previously tuberculous from material brought by means of the portal vein from tuberculous intestines and peritoneum. Florand reported a case that had been jaundiced for two months. A diagnosis of obstruction of the gall duct by a calculus or neoplasm was made. Operation was performed, but the patient died of hemorrhage from the portal vein, due to injury in trying to remove a bunch of tuberculous glands that extended from the duodenum to the portal fissure of the liver. Hodenpyl reported a case of a colored man who had jaundice which the autopsy showed was caused by occlusion of the common duct by a mass of tuberculous lymph glands. Knight reported a case of a child, aged six years, who had increasing jaundice up to the day of death. The autopsy here also showed a mass of tuberculous lymph glands the size of a walnut in the transverse fissure of the liver and around the hepatic duct and portal veins. Köster reported a case of a three-year-old boy who had been jaundiced for two years. A little while before death the jaundice disappeared. At the autopsy there was found in the portal fissure a tuberculous gland that had become caseous and opened into the common bile duct.

The intrahepatic, toxemic, or hemohepatogenous cases of jaundice occur in those cases where there is tuberculosis of the liver itself. Tuberculosis of the the liver may manifest itself in three ways: (1) miliary tubercles, (2) solitary tubercles, and (3) tuberculous cirrhosis. It is only in the first two forms of tuberculosis of the liver that any cases of jaundice have been reported.

Miliary tubercles of the liver are of frequent occurrence. Arnold considered them almost a constant finding in cases of tuberculosis. Rosenberger found miliary tubercles in 83.8 per cent. of livers examined histologically. Simmond found them in 76 per cent. in adults, in 92 per cent. in children, and in 82 per cent. in general. Ullom in examining histologically 100 livers found miliary tubercles in 79. Rolleston puts the occurrence of miliary hepatic tubercles in fatal cases of ordinary pulmonary tuberculosis lower than 50 per cent. Miliary tubercles are found in the liver in cases of general miliary tuberculosis; in these cases the infection is carried by the hepatic artery; or they are found in cases of tuberculosis of the intestines and peritoneum, and in these cases the infection is carried by the portal vein. Ullom in the histological examination of 79 livers containing miliary tubercles found the position of the tubercles to be periportal 40 times, intra-acinal 17 times, and both periportal and intra-acinal 22 times. In 45 cases, examined especially to see the relationship between intestinal ulceration and miliary tuberculosis of the liver, 38 cases showed intestinal ulceration. In these 38 cases the tubercles were found periportal 26 times, intra-acinal 3 times, and both periportal and intra-acinal 9 times.

Although miliary tuberculosis of the liver is such a common finding in tuberculous cases, jaundice caused by it is rare. Rolleston states it to be so rare that it is a curiosity. In 570 autopsies performed upon patients dying of chronic pulmonary tuberculosis at the Henry Phipps Institute, there has been only 1 case of jaundice caused by miliary tuberculosis of the liver.

CASE 2252.—A white man, single, aged twenty years, was admitted to the wards on March 31, 1904. Both he and his father had been hard drinkers, and his father had died of phthisis three years before. He had been sick for six weeks before admission, having been treated at another hospital for typhoid fever. He complained of slight cough, moderate expectoration of a greenish-yellow color, constipation, and night sweats. Examination showed advanced disease of both lungs. The heart had a diffuse pulsation over the whole precordium. The second pulmonic was accentuated. No murmurs were heard, and the dulness was normal. The liver extended two finger breadths below the costal margin. The spleen was palpable one and a half finger breadths below the costal margin. The abdomen was distended. On May 5, the patient was slightly jaundiced, not pronounced, simply an icteroid hue. There was marked tenderness over the liver. By May 14 the icteroid hue was less marked. The patient died on June 24.

The autopsy showed tuberculosis of the lungs, with cavity formation, miliary tubercles, edema, pneumonia, and bronchiectasis; parenchymatous nephritis; enlarged mesenteric glands; small ulcer in the appendix; adhesive pleurisy; fatty liver; heart slightly enlarged, valves normal. The liver was enlarged, weighing, 1860 grams. It was of firm consistence, dark red in color, and had rounded edges. Microscopic examination showed a marked grade of congestion, with distention of the capillaries and characteristic miliary tubercles, with large giant cells, epithelioid and round cells, and caseation.

The other type of tuberculosis of the liver in which jaundice manifests itself is the solitary tubercle, which is classified in three ways: (1) conglomerate tubercle, (2) tuberculous cavities or bile duct tuberculosis, and (3) tuberculous abscess. Conglomerate tubercles are comparatively large caseous masses found in the liver. W. Hale White, discussing this condition, says that before these caseating masses break down they may, unless care is taken, be mistaken for nodules of lymphadenoma. These conglomerate tubercles undergo caseation, soften, and at times, when situated near the bile ducts, rupture into them leaving small cavities in the liver. Solitary tubercles, in whatever form they appear, are an extrenely rare type of tuberculosis of the liver. The writer has been unable to find in the literature a single case of conglomerate tubercles of the liver that showed jaundice. However, two such cases have occurred at the Phipps Institute.

Case 2662.—A white woman, aged forty years, was admitted to the wards of the Phipps Institute August 8, 1904. Her father died at the age of forty-five years of tuberculosis. Mother died at the age of thirty years in confinement. She had one brother and one sister living and well. Two sisters died young of some unknown cause. She had never been sick, but gave a history of excessive use of alcohol over a long period. In the last two years she had lost sixty pounds, and for the past six months had had a cough, worse at night, accompanied by a profuse yellow expectoration. She complained of pain in the lower anterior part of her right chest; dyspnea on exertion, attacks of hoarseness, lasting several days at a time; poor appetite; epigastric pain; distress after eating; constipation; passing blood in the stools; night sweats; slight edema of the ankles. Examination on admission showed an emaciated body, with a face of fairly good color. The heart was normal as well as the abdomen. The lungs showed infiltration of the upper part of the right upper lobe; consolidation, with some softening of the left upper lobe. On August 25 the liver was found slightly enlarged, edges hard, but smooth and not tender. On September 21 the patient was markedly jaundiced; the liver was slightly enlarged. Pressure in the epigastrium caused a great deal of pain. No mass could be felt. On September 26 blood examination showed erythrocytes, 3,840,000; leukocytes, 10,000. The blood was pale and coagulated slowly. The jaundice had become more marked. The conjunctivæ were of a bright yellow color. The whole body was now deeply jaundiced, and pain in the epigastric region was more marked. The liver was distinctly palpable, and the edges sharp and hard. The enlargement was more apparent in the left lobe. On October 1 the jaundice was intense, the abdomen flabby, and distended. There was some free fluid in the peritoneal cavity. Several purpuric spots appeared on the abdomen. From this time on the patient rapidly grew weaker, the jaundice being extreme, pain marked in the epigastrium, sordes on teeth and tongue, pulse weak and irregular, and she finlly died on October 7. The temperature varied between 98° and 101° to 103° until fifteen days before death, when it dropped below normal and so remained until the end. The pulse was between 100 and 120 to 130, and respiration ranged from 27 to 40.

The autopsy on October 8 showed ulcerated tuberculosis of the left lung, tuberculous pneumonia and edema of the right lung, miliary tuberculosis of the spleen, fatty infiltration of the heart, fatty kidneys, with tubercles. The peritoneum contained about 1000 c.c. of yellow, slightly turbid fluid. The liver was enlarged and weighed 2016 grams; dense and granular on the surface; edges were rounded; its color was pale; scattered throughout were numerous small yellowish-white areas of firm consistence, some few were coalesced. The bile duct was patulous. The gall-bladder was distended with fluid, glycerin-like in consistency. Microscopic appearance: Many caseous areas, containing no giant cells and surrounded by epithelioid and small round cells; there were also typical miliary tubercles containing giant cells. The caseous nodules corresponded to the yellowish-white nodules seen macroscopically, and they contained tubercle bacilli in their periphery. There was an increase in the amount of fibrous tissue.

CASE 9808.—A white woman, single, aged forty-five years, was admitted to the wards of the Henry Phipps Institute August 19, 1911. Her father died at the age of sixty years of heart disease and her mother at the age of forty-five years, of dropsy. Four sisters died in childhood of unknown causes. She had two brothers living and well. In 1908 she had had an attack of typhoid fever, but otherwise her health had been good. She had lost about thirty pounds in the past four or five years, and for the past year had had a cough, worse at night, which was accompanied with profuse yellowish sputum. For the past year she had been dyspneic. Her appetite for some months had been poor, and occasionally she vomited. There had been constipation for the past eight months. She had had night sweats nearly every night for the past year. Examination showed advanced disease of both lungs and the outline of cardiac dulness enlarged. The liver was enlarged and extended below the costal margin. On January 23, 1912, there was noticed jaundice in the conjunctivæ. The jaundice increased, and in two days was marked over the whole body. The liver was markedly enlarged, reaching to the umbilicus. It was quite tender. The stools were loose and of a natural color. The patient was nervous and not clear mentally, She did not answer questions intelligently. The jaundice lessened a little until January 29, when it became a little more intense and then gradually decreased until February 10, the day before her death, when it became markedly increased. All this time the patient had a slight talkative delirium, with some delusions. The liver remained enlarged and tender, and at no time was bile found in the urine or feces. The temperature varied between 95° and 103°, the pulse between 90 and 120, and the respiration between 18 and 36.

The autopsy showed chronic pulmonary tuberculosis, chronic adhesive pleurisy, chronic tuberculous laryngitis, chronic pericarditis, with effusion, tuberculous enteritis and colitis, congenital cystic kidneys, and congestion of the spleen. The liver was enlarged, weighing 2300 grams. It was yellowish-brown in color, firm in consistency, and had well-marked, rather sharp edges. On section it was yellowish-brown in color, and there were seen numerous small yellow areas, fairly well-outlined, about 1.5 to 2 cm. in diameter, arranged in groups of four or five. Scattered in many places, closer and more numerous, were smaller areas about 1 mm. in diameter. They were poorly defined, raised above the surface, translucent, and gray. In many places the interlobular septum was thickened. Iodine test for amyloid was positive. The gall duct was patulous. The gall-bladder was normal in size, and contained a dirty reddish-brown, thick fluid.

Rolleston in discussing bile-duct tuberculosis states it is remarkable that jaundice is constantly absent, inasmuch as there is definite obstruction in at any rate some of the bile ducts. Bristowe found 12 cases of small tuberculous cavities of the liver in 167 cases of tuberculous ulceration of the intestines, but he makes no statement whether or not jaundice was present in any of them. Thayer has reported a case of tuberculous cavities of the liver which showed jaundice for a week, but cleared up some time before death. Warthin had a case of bile-duct tuberculosis which had intense jaundice for three months before death.

At times the solitary tubercles become secondarily infected with pyogenic microörganisms, soften down, and form abscesses. Mayo-Robson has reported a case of tuberculous abscess of the liver which was successfully operated on and made a complete recovery. He states that for three months prior to the operation the patient had a slight tendency to jaundice.

That jaundice is a rare condition in tuberculosis is admitted by almost all who have had any experience in tuberculosis. Warthin has had a most unusual experience. He states that 80 per cent. of his cases of acute general miliary tuberculosis of the typhoid form have shown a well-defined icterus, either slight, moderate, or well-marked in grade, and about the same percentage of cases of chronic tuberculosis ending in a general miliary metastasis have also shown the same occurrence of jaundice. The writer's experience at the Phipps Institute dealing with chronic pulmonary tuberculosis for the most part had been quite different. In 570 cases that have come to autopsy in that institution, there has been but 3 cases of icterus; the findings in these cases are reported above. In addition to these 3 cases that died in the institute there were 4 other cases (Nos, 379, 1155, 3495 and 4748) that have shown jaundice; 3 of them showed a slight grade of jaundice and 1 a distinct icterus. These 4 cases left the hospital before death, and, of course, we could not prove that the jaundice was due to a tuberculous condition of the liver. These 7 cases of jaundice are all that have occurred in 1748 cases that have been in the wards of the hospital from the time of its inception until February 1, 1912.

From such a small number of cases it is impossible to draw any conclusions, but taken in connection with cases reported in the literature they are suggestive: (1) Jaundice occurring in the course of chronic tuberculosis is probably due to some form of tuberculosis of the liver, either extrahepatic or intrahepatic. (2) The grade of the jaundice is helpful in surmising the pathological condition, intense jaundice being probably due either to pressure of tuberculous glands on the extrahepatic ducts or to some form of the solitary tubercles of the liver; milder forms being produced by a miliary tuberculosis of the liver.

REFERENCES.

1. Rolleston, H. D. Diseases of Liver, Gall-bladder, and Bile Ducts, 1905.
2. Florand, M. La Semaine Médicale, 1899, p. 20.
3. Hodenpyl. Medical Record, 1898.
4. Knight. Quar. Med. Jour., Sheffield, July, 1895.
5. Köster, A. Centralbl. f. inn. Med., 1896, S. 213.
6. Arnold. Quoted from Ullom.
7. Rosenberger. Trans. Path. Soc. Phila., ix, 1906.
8. Simmond. Deutch. Arch. f. klin. Med., 1880, Band xxviii, 450.
9. Ullom. Proceedings of the Sixth International Congress on Tuberculosis, vol. I, Part I, 363.
10. Hale White, W. Common Affections of the Liver, 1908.
11. Bristowe, J. S. Transactions Path. Soc., London, ix, 241
12. Thayer. Johns Hopkins Hos. Bull., May, 1911.
13. Warthin. International clinics, vol. I, 21st series, 1911.
14. Mayo-Robson. Clin. Soc. Trans., London. 1895, xxviii, 83
15. Maylard, A. E. Abdominal Tuberculosis.
16. Wethered, F. J. Trans. Path. Soc., London, xl, 139.
17. Graham, J. E. American System of Practical Medicine, vol. III.
18. Murchison. Diseases of the liver, 3d edition, p. 279.

HYALINE DEGENERATION OF THE ISLANDS OF LANGERHANS IN NON-DIABETIC CONDITIONS.

By Russell L. Cecil, M.D.,

ASSISTANT PHYSICIAN TO THE PRESBYTERIAN HOSPITAL; INSTRUCTOR IN CLINICAL MEDICINE IN THE COLUMBIA UNIVERSITY MEDICAL SCHOOL, NEW YORK.

(From the Pathological Laboratory and the Medical Services of the Presbyterian Hospital, Columbia University, New York.)

Hyaline degeneration of the islands of Langerhans was first described in 1900 by Opie,[1] who noted its association with diabetes mellitus. Since then numerous observers have encountered hyaline islands in diabetes, and the condition is now recognized as a common finding in this disease.

In a series of 90 cases of diabetes which I[2] studied in 1909, sclerosis of the islands was noted in 60, and hyaline degeneration

---

[1] Jour. Exp. Med., 1900 to 1901, v, 397, 527.

[2] Cecil, Jour. Exp. Med., 1909, 266; 1911, xiv, 500.